Review Article

Urinalysis - The Liquid Kidney Biopsy

Abstract

Urinanalysis, considered the "liquid biopsy" of the kidney, is rapidly becoming a lost art among nephrologists. A well-performed urinanalysis provides information on the presence of kidney disease, identifies the affected compartment, and helps monitor disease activity. The three main components of urine analysis are gross examination, dipstick tests, and urine sediment examination under microscopy. The urine sediments should be analyzed under a bright-field, dark-field, phase-contrast, and polarization microscope for better visualization and categorization of cells, casts, and crystals. The Sternheimer-Malbin (SM) stain can enhance urine sediments under a microscope. The urine sediment, like in a kidney biopsy, should be systematically analyzed for evidence of glomerular (dysmorphic RBCs, acanthocytes, RBC casts, lipid casts) and tubulointerstitial (RTE cell casts, Granular casts, Leucocyte casts, broad waxy casts) injury. For crystal identification in sediments, knowledge of urinary pH, morphology, and birefringence features under polarized light is essential.

Keywords: *Acanthocytes, Casts, Crystals, Urine microscopy, Urine sediment examination*

Sabarinath Shanmugam[1], Mythri Shankar[2], Jay R Seltzer[3], Urmila Anandh[4]

[1]Department of Nephrology, Jawaharlal Institute of Postgraduate Medical Education and Research, Puducherry, [2]Department of Nephrology, Institute of Nephro-Urology, Victoria Hospital Campus, Bangalore, India, [3]Department of Nephrology, Missouri Baptist Medical Center, Saint Louis, United States, [4]Department of Nephrology, Amrita Institute of Medical Sciences and Research Centre, Faridabad, Haryana, India

Corresponding author:
Sabarinath Shanmugam, Department of Nephrology, Jawaharlal Institute of Postgraduate Medical Education and Research, Puducherry, India. E-mail: sabarivenus@gmail.com

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Introduction

Urine analysis (Urinalysis) is a non-invasive, easily accessible diagnostic tool for nephrologists and clinicians. It is considered the "liquid biopsy" of the kidney. Urinanalysis is suggested for almost every patient visiting a nephrologist. Therefore, thorough knowledge of the interpretation of urine analysis is essential. This review focuses on the basics of urine analysis, techniques, and stains that can help better visualize urine sediments.

How to Collect Urine Specimens?

Specimen collection

Urine should be collected in a clean, dry, sterile container; it should be free of preservatives or additives unless specified by the testing protocol. Before collection, the patients should be informed about the "clean-catch" method (washing hands before sample collection; male patients are instructed to retract the foreskin and clean the glans of the penis; females should clean the labia and urethral meatus). Patients must collect a midstream urine sample (15-30 mL) to minimize the chance of contamination from penile/vaginal secretions and microbiota. In patients with indwelling urinary catheters, a sample should be obtained directly from the catheter tubing via the sampling port (after draining any standing urine from the drainage tube into the collection bag) rather than from the urometer or drainage bag to ensure that the sample represents recently produced urine and avoids contamination by debris in the collection bag.

Specimen preservation

Urine is an unstable fluid; the composition changes as soon as it is eliminated from the body. Urine particles can lyse rapidly after collection, especially when pH is alkaline and specific gravity or osmolality is low. It should be examined within 2-4 hours of collection due to the instability of some urine components (cells, casts, and crystals).[1] If not possible, the sample can be refrigerated at 2 to 8°C for up to 24 hours, which slows the decomposition process, and then re-warmed to room temperature before assessment. The changes that occur with time after urine collection are: a) Rise in pH, b) dissolution of cells and casts, c) crystallization of solutes, d) loss of bilirubin and ketone bodies, and e) overgrowth of contaminating microorganisms.

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Urine Examination

A complete urine analysis has three components: a) Gross examination, b) Dipstick analysis, and c) microscopic examination.

Gross Examination

Color

Under normal conditions, the color of urine ranges from pale to dark yellow and amber. The main causes of abnormal urine colors have been listed in Figure 1. Red or red-brown urine is noticed in various conditions like hematuria, hemoglobinuria, myoglobinuria, or due to certain drugs like rifampicin. Finding the cause is of utmost importance clinically. The stepwise approach to red or red-brown urine is;

Step 1: Centrifuge the urine

Step 2: Observe the color of the supernatant

If the supernatant is clear and only the sediments are red colored, likely hematuria.

Step 3: If the supernatant is red, do a dipstick on the supernatant for heme.

If negative, look for other causes like medication intake (Phenytoin, rifampicin, or hydrocobalamine), consumption of beets (beeturia), and rarely, acute intermittent porphyria.

If positive, it could be due to either hemoglobinuria or myoglobinuria.

Step 4: Look for the plasma color to differentiate between myoglobinuria and hemoglobinuria. If clear, it is myoglobinuria; if red, it is hemoglobinuria.

Myoglobin is a monomer with a molecular weight of 17 kDa and is not protein-bound. As a result, it is rapidly filtered and excreted, allowing the plasma to retain its normal color. Hemoglobin exists in tetramers (69 kDa) and dimers (34 kDa), making it difficult for the glomerulus to filter. It is protein-bound (with haptoglobin), and unbound dimers are filtered only after the haptoglobins are fully saturated, thus retaining the red color in plasma in case of hemoglobinuria.

Turbidity

Normally, urine is transparent. Turbidity is indicative of an increase in concentration of any urine particles (like crystals). Infection and contamination with genital secretions frequently cause turbidity.

Odor

Infected urine can have an abnormal, pungent odor, caused by the production of ammonia by bacteria. Other well-described odors of urine have been listed in Figure 2.

Foaminess

Foamy urine upon voiding is considered a sign of proteinuria. A single layer of large bubbles that quickly disappears upon urination can be considered normal. The appearance and persistence of multiple layers of small to medium bubbles in urine voided into a container needs evaluation. The presence of significant protein acts as a surfactant and forms foam. Besides protein, aminoaciduria (Fanconi syndrome) can also cause significant foaminess in urine [Figure 3].

Urine Dipstick analysis

The urine dipstick provides a semi-quantitative assessment of urine characteristics on a series of colorimetric pads

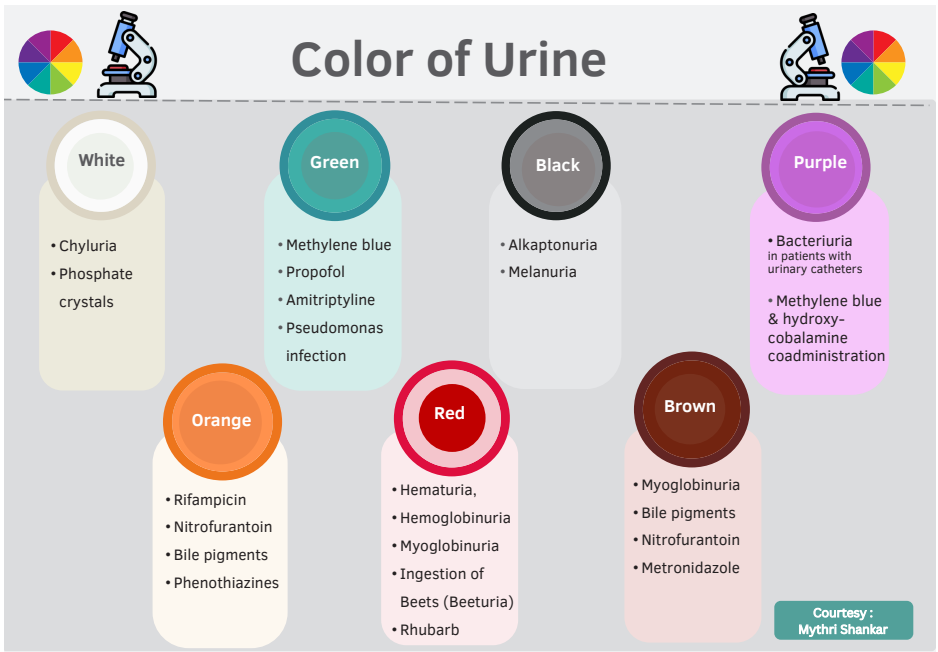

Figure 1: Color of urine and its significance.

embedded on a test strip. The following parameters are analyzed:

Urine pH

The pH reflects the degree of urinary acidification. The physiological pH of urine ranges between 4.5 and 8. In routine practice, it is measured using the dipstick. However, a pH meter is needed for accurate measurement. The appropriate kidney response to acidemia is to increase urinary acid excretion, resulting in a pH level below 5. Urinary pH generally reflects the serum pH, except in patients with renal tubular acidosis (RTA). Infection with any pathogen that produces urease can result in a urine pH >8, even if urinary acidification is normal.

Hemoglobin

Detection of hemoglobin using a dipstick has 95% to 100% sensitivity (even detects 1-2 RBCs/hpf)[2] and 65% to 93% specificity.[3] Heme acts as a pseudoperoxidase. A color change happens when heme-containing urine is exposed to a strip containing peroxide and chromogen. A positive dipstick may indicate hematuria (presence of intact RBCs), hemoglobinuria, or myoglobinuria. Confirmation of hematuria requires urine microscopy. A high concentration of pseudo-peroxidase-containing bacteria (*Enterobacteriaceae*, *Staphylococci*, and *Streptococci* species) can result in false positivity.

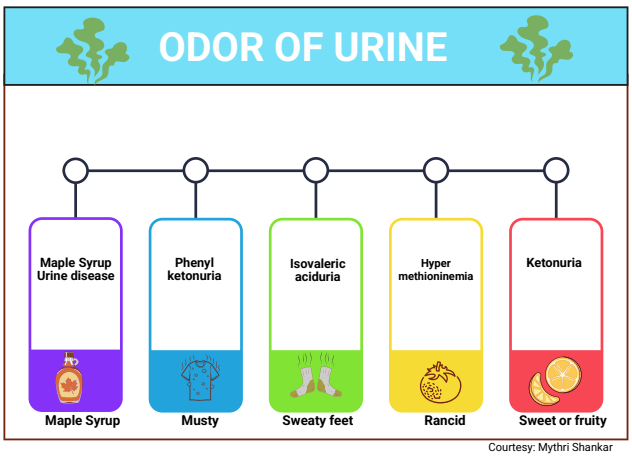

Figure 2: Odor of urine.

Figure 3: The typical foamy urine denoting significant proteinuria and disappearance of foaminess on remission.

Leucocyte esterase

This evaluates the presence of leucocytes in urine. Lysed neutrophils and macrophages release indoxyl esterase, which the dipstick detects. Proteinuria and glucosuria may lead to a false-negative test for leukocyte esterase.

Nitrite

Urinary tract infection due to nitrate reductase-expressing bacteria (*Enterobacteriaceae*) that convert urinary nitrate to nitrite may yield a positive test. By contrast, urinary infections with *Enterococcal* species expressing low levels of nitrate reductase may test negative for nitrites. A negative test does not rule out a urinary tract infection (UTI).

Specific gravity

Urinary specific gravity (SG) is the ratio between the density of urine and the density of an equal volume of pure distilled water. Specific gravity and urinary osmolality are well associated. The urine SG generally rises by approximately 0.001 for every 35 to 40 mOsmol/kg increase in urine osmolality. A urine osmolality of 280 mOsm/kg is (similar to plasma osmolality) associated with a urinary specific gravity of 1.010. This is observed when there is an impairment in the urinary concentration capacity of the kidney, such as acute tubular necrosis and CKD.

SG, evaluated by a reagent strip, measures the *ionic concentration* of urine. Urine pH>6.5 underestimates the SG, whereas overestimation is noted when the protein concentration is >7.0 g/L. Hence, refractometry is an ideal technique for measuring SG.

Protein

In normal conditions, the glomerular capillary wall is permeable to <20 kDa. Most of the small fraction of filtered protein is reabsorbed by the tubules. Hence, proteins are normally present in trace amounts (<150 mg/day). Among them, 1/3 is albumin, 1/3 is Tamm-Horsfall glycoprotein, and the rest comprises other plasma proteins.

The urinary dipstick is more sensitive to albumin (detection limit: 0.25 to 0.30 g/L) and insensitive to non-albumin proteins like immunoglobulins. Hence, the positivity indicates glomerular proteinuria. This strip test is based on the effect of albumin on a buffer (*tetrabromophenol blue*), which causes a change in pH proportional to the albumin concentration. The pad changes its color from pale green to green and blue according to the pH changes induced by albumin. This strip test can produce false-positive results in the following situations.

a. Highly alkaline urinary pH (pH>8)
b. After the use of iodinated contrast[4]
c. Presence of gross hematuria[5]
d. When specific antiseptics (e.g., chlorhexidine, benzalkonium) are used for clean-catch urine samples[6]

A dilute urine may underestimate the degree of albuminuria. The reagent strip supplies only a semiquantitative urine albumin assessment, expressed on a scale from 0 to +++ or ++++ [Table 1].

Glucose

With a dipstick using glucose oxidase, glucose is first oxidized to gluconic acid and hydrogen peroxide. Then, through the catalyzing activity of peroxidase, the latter reacts with a reduced colorless chromogen to form a colored product. This test detects a concentration of 0.5 to 20 g/L. False-negative results can occur in the presence of bacteria and ascorbic acid; false-positive findings are observed with a very acidic urinary pH and in the presence of oxidizing detergents that oxidize the chromogen independently of hydrogen peroxide formation. Glycosuria can be due to the inability of the kidney to reabsorb filtered glucose in the proximal tubule despite normal plasma glucose concentration or glucose excretion related to high plasma glucose concentrations overwhelming the capacity of the renal tubules to reabsorb glucose. In patients with normal kidney function, significant glycosuria occurs when the plasma glucose concentration is >180 mg/dL.

Ketones

Reagent strips do not detect β-hydroxy-butyric acid; only acetoacetic acid and acetones are detected. This dipstick is based on the reaction of nitroprusside with acetoacetate and acetone. Ketones are detected in urine in states like diabetic acidosis, fasting, vomiting, or strenuous exercise.

Microscopic Sediment Examination

Microscopy is an integral in urine analysis. It can be manual or automated, using analyzers. Preparing the urine for sediment examination is essential in urine microscopy.[7]

For preparing the sample, 10-15 mL of urine is centrifuged at 400g for 10 minutes. The supernatant is then discarded. The pellet is then resuspended by gently shaking the tube. A pipette is used to place the resuspended pellet on a glass-slide and the coverslip is placed on top. To correctly interpret the findings, the sample's pH and specific gravity must be determined. Alkaline pH and/or low specific gravity, especially <1.010, favor erythrocyte and leucocyte lysis, which can give false-negative results on microscopy.

Table 1: Semiquantitative estimation of protein by dipstick analysis

Dipstick protein reading	Protein excretion (mg/dL)
Negative	<10
Trace	15
1+	30
2+	100
3+	300
4+	>1000

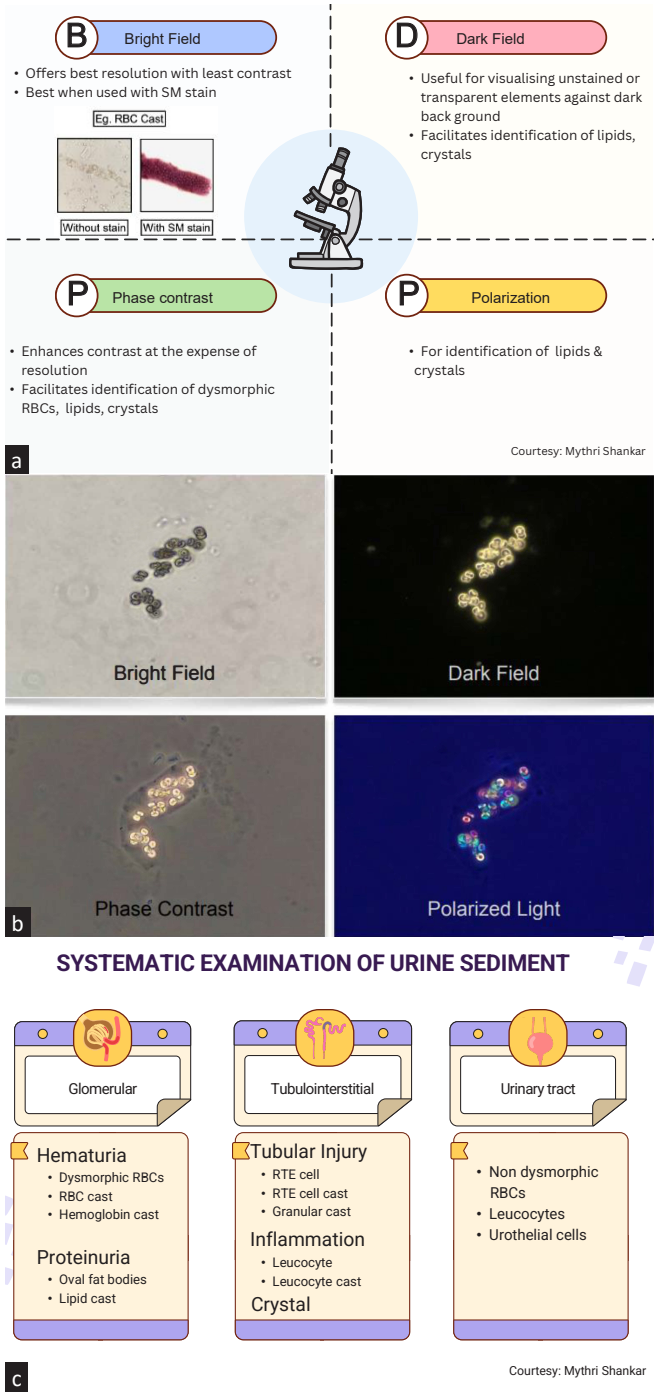

Figure 4: (a) Different microscopic techniques for urine sediment examination, (b) importance of examining the urine sediments by all four techniques, (c) systematic examination of urinary sediment. SM: Sternheimer-Malbin, RTE: Renal tubular epithelial cells.

The knowledge of pH is also useful for accurately identifying crystals.

Stains used:

- Add one drop of Sternheimer-Malbin (SM) to the pellet before transferring it to the glass slide and wait 1 minute for uptake.

- Add 3-5 drops of Sudan III stain to the pellet to better identify the lipids.

What microscope to use?

The different microscopic techniques used to analyse urine sediments have been listed below [Figure 4a].

The importance of using all four techniques!

It is often helpful to utilize all available modalities. One of the best examples has been given below [Figure 4b]. In the bright field image, circular biconcave objects are visible. The objects appear brighter under the dark field microscope due to their high refractive index (typical of lipids and crystals). Phase contrast microscopy denotes that they can be observed within the cast. The protein matrix of the cast, which is not visible in the bright field, is well seen in phase contrast. Still, it is difficult to comment whether it is RBC, lipid droplets, or crystals. The polarized image shows birefringent, and hence, they can be detected as crystals. So, what is not obvious in bright field can be determined as crystals in polarized microscopy.

Automated or manual technique?

Although automated urine analysis systems are time-saving, cost-effective, and standardized, they may be inadequate for identifying and classifying sediment particles, such as casts and crystals, in highly pathological samples.[8,9] It is well demonstrated that the diagnostic yield of urine sediment examination performed by a trained clinician is substantially higher than that done by laboratory staff.[10]

Systematic approach to urine sediment examination

The urine, like a renal pathology specimen, can be analyzed systematically, as shown in Figure 4c.

Glomerular injury

Dysmorphic RBCs

Distinguishing glomerular hematuria from non-glomerular hematuria is of utmost clinical importance. Phase contrast microscopy is superior to bright field in assessing RBC morphology. Isomorphic RBCs are 6 µm in diameter and resemble those in peripheral smear. Any that are not isomorphic are labelled as dysmorphic RBCs. Isomorphic

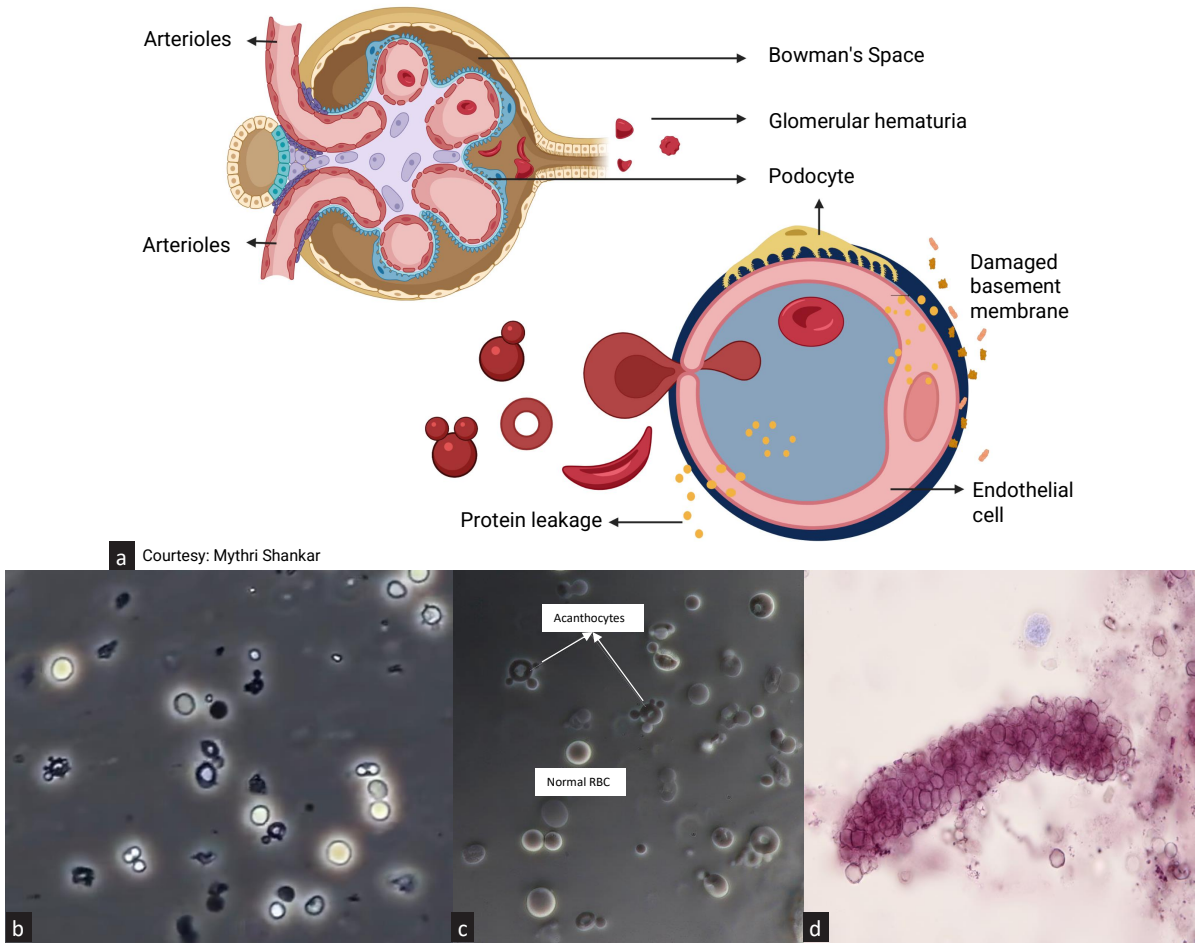

Figure 5: (a) The average diameter of an erythrocyte is 6–8 micrometres, which is about 100 times larger than the endothelial fenestrations (60–80 nanometres) in the glomerulus. Red blood cell (RBC) diapedesis occurs through a disrupted glomerular basement membrane, leading to membrane protrusions in the RBCs. The differential pH, osmotic forces in the tubular fluid, and proteases secreted by inflammatory cells further alter the RBC membrane. (b) Phase contrast microscopy showing dysmorphic RBCs and Acanthocytes, x1000; (c) Dark field illumination, showing an acanthocyte, x1000; (d) RBC cast with SM stain, x400. (Image Courtesy: Jay Seltzer).

RBCs can be seen in any cause of hematuria, whereas the presence of dysmorphic RBCs indicates glomerular origin.

How are dysmorphic RBCs formed?

Dysmorphic RBCs may have different shapes, such as ring shapes, single or multiple blebs, or protrusions. Due to the membrane loss, these cells are typically smaller (3 µm) than isomorphic RBCs. The RBCs must pass through gaps in the injured GBM, and are exposed to an osmotic gradient across the tubules and also acidic urine, which makes their appearance dysmorphic [Figure 5a].

Acanthocytes and their importance

Acanthocytes are a subset of dysmorphic RBCs (which have membrane protrusions) that have a 98% specificity and 52% sensitivity for diagnosing glomerular hematuria [Figure 5b and c]. Though there is no general agreement, dysmorphic RBCs >40%, ≥5% acanthocytes, or one or more red blood cell casts/50 lpf is a good criterion to diagnose glomerular hematuria.[11]

RBC casts

Casts are cylindrical structures formed in the tubular lumen. The basic architecture is formed by Tamm-Horsfall mucoprotein (uromodulin). The presence of RBC casts suggests glomerular hematuria [Figure 5d]. Casts are best identified at the edge of the coverslip.

It is worth noting that RBC casts are not exclusive to glomerulonephritis. Patients (~30%) with acute interstitial nephritis can have RBC casts. This is likely due to the RBCs that enter the tubules from the inflamed interstitium and form the cast.

Lipid casts

This is seen generally in nephrotic syndrome. The fat droplets can be seen within the tubular cells (oval fat bodies) or as a cast (lipid cast). The fat droplets show a characteristic Maltese-cross appearance in polarized light. In healthy individuals, lipoprotein filtration is minimal. Lipoprotein-bound cholesterol, especially high-density lipoprotein, gets filtered in patients with nephrotic syndrome. The proximal tubular cells take up this filtered lipoprotein, and when the tubular cells get desquamated, oval fat bodies are seen in urine [Figure 6a-c]. Lipid casts were also noted in autosomal dominant polycystic kidney disease.

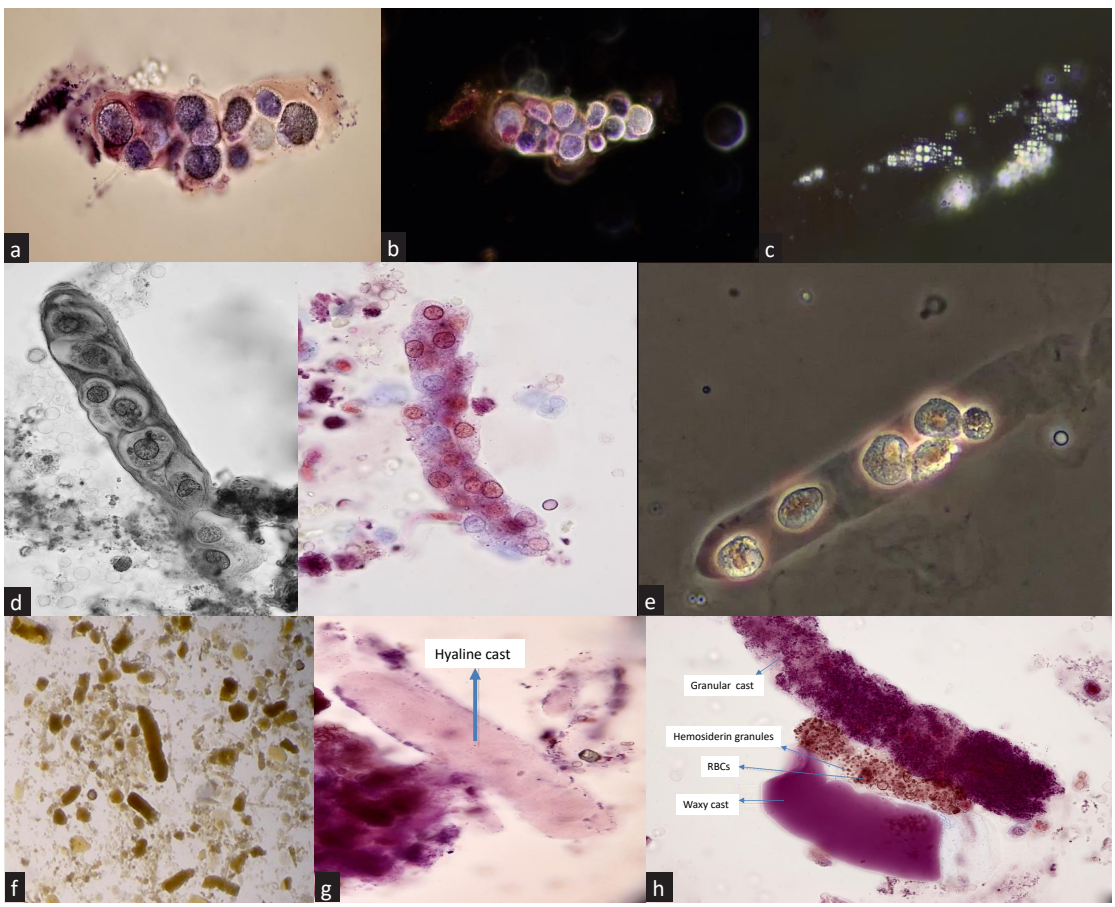

Figure 6: (a) Oval fat bodies with SM - stain (Bright field), x1000; (b) Oval fat bodies (Dark field), x1000 (c) Oval fat bodies with Maltese cross appearance on polarised microscopy, x1000; (d) Renal Tubular epithelial cell (RTEC) cast - brightfield with SM stain (converted to grayscale) & Cluster of tubular epithelial cells - brightfield with SM stain, x1000; (e) RTEC casts (phase contrast), x1000 (f) Granular casts, x1000; (g) Waxy Hyaline casts, x1000; (h) Hyaline cast, RBC cast and Granular casts with SM stain, x1000. (Image Courtesy: Jay Seltzer).

Tubulointerstitial injury

RTE Cell casts

Renal Tubular Epithelial cell (RTE) casts are observed when tubular epithelial cells are desquamated. Urinary RTE cells (RTEC) [Figure 6d and e] can be oval, round, polygonal, or columnar and typically have a high nucleolar-to-cell diameter ratio. An RTEC will be approximately double the size of an RBC. More severe tubular injury increases the number of RTECs and RTE casts observed on sediment examination. It is best visualized with a bright field microscopy under SM stain.

Granular casts

The granular casts may be fine, coarse, or mixed, generally reflecting tubular injury. These granularities are due to particles from degenerated RTECs (seen as granules in electron microscopy), admixed with uromodulin. Also, it has been shown that filtered plasma proteins in pathologic states can get stuck with the uromodulin, giving a granular appearance. In general, scattered fine granular casts do not have a significant clinical value, but the abundance of coarsely granular casts, i.e., "muddy brown" granular casts (>10/lpf), is typically characteristic of acute tubular injury [Figure 6f]. The mitochondrial pigments, or lipofuscin, are responsible for the dark brown color of the muddy brown granular casts.[12]

Leucocytes and leucocyte casts

Urinary neutrophils are commonly associated with infection. If urine culture is negative, evaluation should be done to rule out acute interstitial nephritis, renal tuberculosis and nephrolithiasis. Urine for eosinophils can be detected by using Wright or Hansel stain. Though urinary eosinophils are traditionally considered a marker

Table 2: Showing crystals, their appearance, and associated conditions

Crystals	Appearance	Urine pH	Birefringence	Associated conditions
Calcium oxalate (monohydrate)	Dumbbell shaped	5.4-6.7	Strong	Nephrolithiasis, ethylene glycol poisoning, and excessive oxalate absorption
Calcium oxalate (dihydrate)	Envelope shaped		Weak	
Calcium phosphate	Needles of various sizes, prisms	> 7	Strong	Nephrolithiasis
Triple phosphate	Coffin lids	6.2-7	Strong	Infection with urea-splitting organism
Uric acid	Rhomboid	5.4-5.8	Strong Polychromatic	Nephrolithiasis, tumor lysis syndrome
Cystine	Hexagonal shaped	5.5	Weak	Cystinuria
Cholesterol	Thin plates	5.5	Negative	Nephrotic syndrome

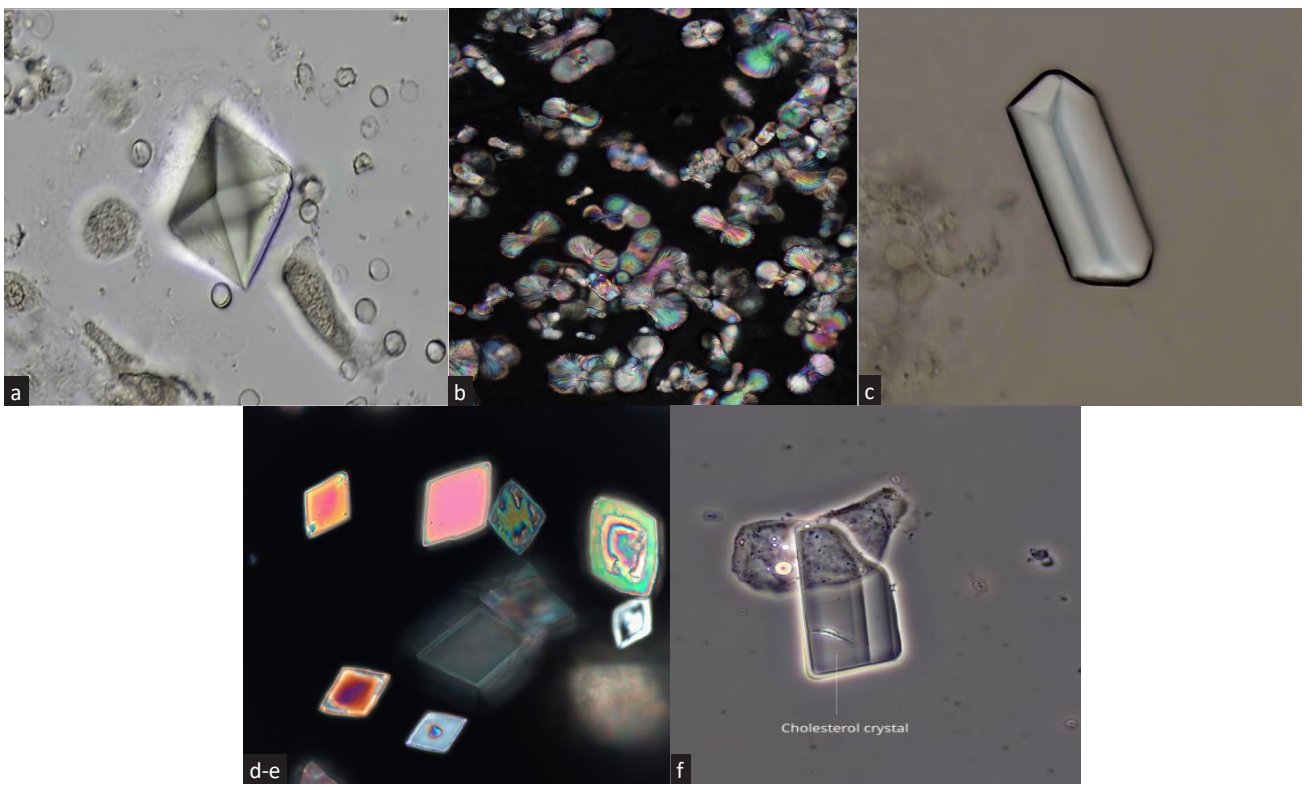

Figure 7: (a) Envelope-shaped Calcium dihydrate crystals, x1000; (b) Dumbbell-shaped calcium monohydrate crystals, x200; (c) Coffin-lid-shaped triple phosphate crystals, x1000; (d) Uric acid crystals, which are hexagonal, x1000 and (e) polychromatic; (f) thin plates of cholesterol crystals, x1000. (Image Courtesy: Jay Seltzer).

of acute interstitial nephritis (AIN), evidence shows that only 34% of patients with AIN had eosinophilluria.[13,14] WBC casts are indicative of interstitial inflammation, and it is important to note that only 3% of patients with biopsy-proven AIN have WBC casts in urine.[15]

Other important considerations in urine analysis:

Hyaline casts

They are generally nonspecific, slightly more refractile than water and have a transparent, empty appearance [Figure 6g and h].

Broad waxy casts

This is typically associated with chronic renal failure. The broadness of the cast is due to their formation in large, dilated tubules with little flow.

Crystals

Knowing urinary pH, morphology, and birefringence features under polarized light allows crystal identification. The commonly noticed crystals are uric acid crystals, calcium oxalate crystals, and calcium phosphate crystals. It is an occasional finding in most instances, as it may reflect transient supersaturation of urine caused by mild dehydration or even precipitation of crystals in the interval between urine collection and urine examination. The persistence of such crystals in repeated samples needs evaluation. Some crystals are always pathogenic, like cholesterol crystals (nephrotic proteinuria), cysteine crystals (cystinuria), and 2,8 dihydroxyamine crystals (deficiency of the enzyme adenine phosphoribosyltransferase).[16] Table 2 shows the different morphologies of crystals, urinary pH, birefringence on polarized light, and associated conditions. The morphology of crystals in urine has been shown in Figure 7.

Recent Advances

The disadvantages of manual microscopy examination are that it is labor-intensive, time-consuming, introduces inter-observer variability, and potential pre-analytical errors. As a progress in the field, automated digital microscopy and flow cytometry have been introduced for enumerating urine particles. The software image analysis of the digital microscope is crucial to automated urine sediment examinations. Automated digital microscopy, based on pattern recognition, produces real images that can be reviewed by experts. Compared with manual microscopy, it shows good performance and agreement in detecting RBCs, WBCs, bacteria, and squamous epithelial cells. However, it is less reliable in detecting non-squamous epithelial cells, non-hyaline casts, unusual crystals, and lipids.[17]

Laser-based Flow cytometry is being investigated as a screening test for pyuria or bacteriuria. The combined analysis of scattered light and fluorescence allows the rapid identification and differentiation of particles such as leukocytes, bacteria, and iso- or dysmorphic RBCs. But, flow cytometric methods make a scattergram, not an image; the laboratorian cannot discard a manual microscope to verify and differentiate complex urinary elements.[17]

To conclude, urine analysis is a fundamental diagnostic tool for nephrologists. Given the sophisticated automated urine analyzer, the number of nephrologists performing urine analysis is declining in the current era. However, it should be remembered that a sediment examination by a trained nephrologist cannot be completely replaced by an automated urine analyzer in diagnosis and management.

Conflicts of interest: There are no conflicts of interest.

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